British Heart Journal, 1972, 34, 1232–1237.

Electrophysiological features of Mobitz type II AV block occurring within the His bundle

Prem K. Gupta, Edgar Lichstein, and Kul D. Chadda

From the Department of Medicine, Division of Cardiology, Mount Sinai Hospital Services, City Hospital Center at Elmhurst, and the Mount Sinai School of Medicine of the City University of New York, U.S.A.

Two patients with second-degree AV block and narrow QRS were studied by His bundle electrography. The first patient had Mobitz type II AV block on the standard electrocardiogram but showed 2:1 AV conduction when His bundle electrograms were recorded. Intravenous atropine increased the atrial rate but 2:1 AV conduction persisted. The block was localized distal to His bundle activity and was thought to be in the distal His bundle segment.

The second patient had 2:1 AV conduction and showed split or double His bundle potentials, i.e. H and H'. Nonconducted atrial depolarizations were blocked distal to the H deflection suggesting that the site of block was within the His bundle. AV conduction improved to 3:2 after intravenous atropine. Atrial pacing performed in this patient during 1:1 AV conduction produced a typical Mobitz type II AV block within the His bundle.

The technique of His bundle electrography, introduced by Scherlag *et al.* (1969), has made it possible to localize the site of delay in various types of atrioventricular (AV) block. Mobitz type II AV block with bundle-branch block is thought to represent failure of conduction in the contralateral bundle (Langendorf and Pick, 1968). Mobitz type II AV block in the absence of bundle-branch block usually indicates a more proximal site of delay, i.e. the AV node (Rosen *et al.*, 1971) or within the His bundle (Narula and Samet, 1970).

The two patients presented in this report had Mobitz type II AV block with narrow QRS complexes. Block in both patients was localized within the His bundle. AV conduction was studied with the use of atropine in both patients and with atrial pacing in one.

Method

His bundle electrograms were obtained by the method described by Scherlag *et al.* (1969), using an Electronics for Medicine multichannel recorder. Electrograms were recorded at a paper speed of 75 and 150 mm/sec and a frequency response of 40–500 cps. One or more leads of the peripheral electrocardiogram were recorded simultaneously. Atrial pacing was performed using a size 4 bipolar semi-floating electrode catheter[1] passed percutaneously via the right subclavian vein into the right atrium. A Model 5840 Medtronic pacemaker was used and the pacing rate was increased gradually from 80 to 150/minute.

Case 1

A 72-year-old white woman was admitted to hospital when a routine physical examination revealed a pulse rate of 40 a minute. The patient denied any history of syncope or dizziness. Hypertension was noted 4 years ago and was treated with oral diuretics. She complained of dyspnoea after walking one block and occasionally needed more than two pillows.

On admission the patient was in no distress. Pulse rate was 74 a minute, with occasional dropped beats. Blood pressure was 190/100 mmHg in both arms. The neck veins were slightly distended at 45°. Examination of chest revealed bilateral basilar râles. The point of maximal cardiac impulse was not palpable. There was a grade 2/6 holosystolic murmur at the mitral area which radiated to the axilla. 1+ pitting oedema of the ankles was noted. Electrocardiogram on admission (Fig. 1) showed sinus arrhythmia with sinus rate varying between 72 and 80 a minute. PR interval was 0·20 sec and the QRS duration measured 0·09 sec. The QRS morphology was consistent with left anterior hemiblock. Nonconducted P waves were seen without any change in the preceding PR interval. Chest *x*-ray

Received 14 April 1972.

[1] Electro-Catheter Corporation, Rahway, New Jersey.

showed left ventricular enlargement with no pulmonary vascular congestion. Laboratory data included normal haematocrit, BUN, fasting blood sugar, cholesterol, and electrolytes. Because of electrocardiographic evidence of Mobitz II AV block, a temporary transvenous pacemaker was inserted and His bundle recordings obtained. After baseline records, the patient was given atropine 0·8 mg intravenously.

Results

During the procedure the patient showed 2:1 AV conduction with atrial rate of 82 a minute and ventricular rate of 41 a minute.

His bundle electrograms are shown in Fig. 2. Each atrial depolarization is followed by a biphasic His bundle potential with an AH interval of 120 msec (normal range 70–140 msec). Ventricular activity follows every other His bundle depolarization. The HQ interval in the conducted beats is 50 msec (normal range 35–55 msec).

Intravenous atropine increased the atrial rate to 110 a minute, but 2:1 AV conduction persisted.

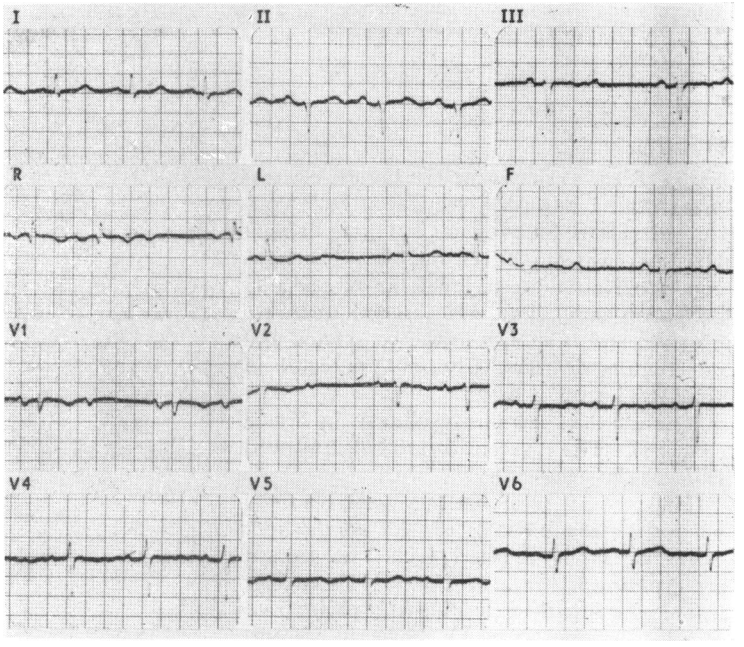


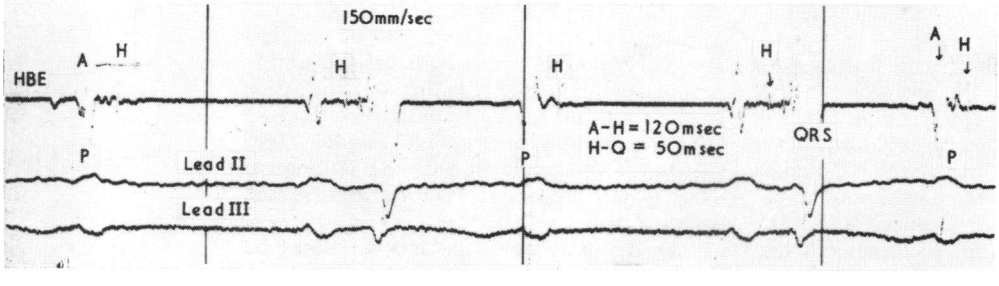
FIG. 1 *Case 1. Electrocardiogram on admission showing left anterior hemiblock and periods of Mobitz type II AV block. PR interval in conducted beats is 0·20 second.*

Case 2

This 57-year-old Negress was admitted to the hospital with increasing shortness of breath and ankle oedema of three days' duration. Two weeks before admission the patient had dizziness and shortness of breath and was noted to have a blood pressure of 200/150 mmHg. She was treated with digoxin, diuretics, and guanethidine. She denied any history of chest pain. On examination the pulse was 45 a minute and blood pressure was 200/90 mmHg in both arms. There was 2+ bilateral ankle oedema. Neck veins were distended at 30°. Examination of the heart revealed the point of maximal impulse in the fifth intercostal space 2 cm to the left of midclavicular line and a short grade 2/6 early systolic murmur at the apex. Bilateral basilar râles were noted. Chest *x*-ray showed left ventricular enlargement and bilateral pulmonary vascular congestion.

Electrocardiogram on admission (Fig. 3) showed sinus rhythm with an atrial rate of 86 a minute and 2:1 AV conduction. The QRS interval was 0·10 sec. There was evidence of left ventricular hypertrophy with strain pattern. Because of the presence of AV block and a history of dizziness, digitalis was discontinued and a temporary transvenous pacemaker was inserted. His bundle recordings were obtained during pacemaker insertion. After baseline recordings, atropine 1 mg was given intravenously. This resulted in transient improvement in AV conduction from 2:1 to 3:2. Demand ventricular pacing was initiated at a rate of 70 a minute and 48 hours later 1:1 AV conduc-

FIG. 2 *Case 1. His bundle electrogram showing 2:1 AV conduction with block distal to the His bundle potential.*



tion returned. At that time atrial pacing was done to study the conduction system under the stress of increased rates.

Results

Fig. 4 (Panel I) shows the His bundle electrogram during 2:1 AV conduction. Each conducted beat is followed by a double or split His bundle potential (H and H'). The nonconducted atrial beats are followed by a H deflection only. The AH interval measures 95 msec and is similar in the conducted and nonconducted beats. The H'Q interval in conducted beats is 35 msec.

The effect of atropine is shown in Fig. 4 (Panel II). The AV conduction has improved from 2:1 to 3:2.

The recordings in Fig. 5A were obtained when the patient was in 1:1 AV conduction. Split His activity is clearly seen. The effect of atrial pacing is shown in Fig. 5B. Pacemaker impulses are marked as (PI). At a pacing rate of 150 a minute, 2:1 AV block is present. Block occurs again distal to the H.

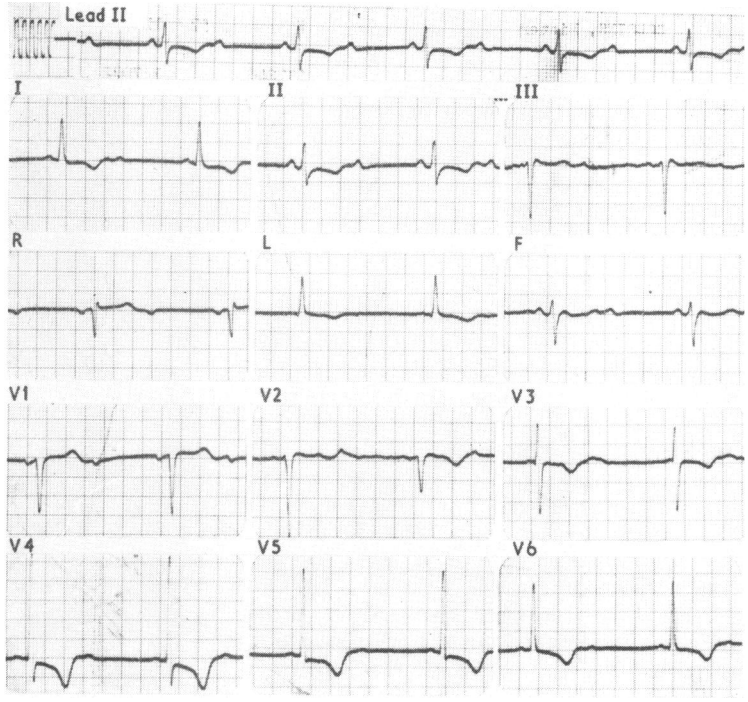


FIG. 3 *Case 2. Electrocardiogram on admission showing 2:1 AV conduction with narrow QRS complexes. The morphology is consistent with left ventricular hypertrophy.*

Discussion

In the majority of patients with Mobitz type II AV block, bundle-branch block is present and the dropped beats represent failure of conduction in the contralateral bundle (Langendorf and Pick, 1968). His bundle electrograms in such cases have localized the block distal to His bundle activity (Narula and Samet, 1970). Two recent reports have described patients with Mobitz type II AV block and normal QRS morphology. In these patients block either occurred at the AV node (Rosen *et al.*, 1971) or within the His bundle (Narula and Samet, 1970). A similar electrocardiogram may also be seen with pseudo type II block where nonconducted His bundle premature impulses cause sudden blocked atrial activity (Rosen, Rahimtoola, and Gunnar, 1970). In a recent experimental study on dog hearts, Spear and Moore (1971) showed that with rapid atrial pacing, Mobitz type II block could be produced within the AV node if the PP interval was shortened by as little as 20 msec.

Narula and Samet (1970) were first to show that Mobitz type II and Wenckebach type AV block may occur within the His bundle. They also described the presence of split or double His potentials. Of 14 cases with infranodal second-degree AV block, they found 4 patients in whom block was localized within the His bundle. Each of these 4 patients had normal QRS morphology as compared to the other 10 who had some form of bundle-branch block.

The 2 cases described in the present report are among the 7 cases of infranodal second-degree AV block studied in our laboratory. The remaining 5 patients had various type of intraventricular conduction abnormalities. This suggests that second-degree AV block within the His bundle may not be as uncommon as previously thought.

Our first patient showed a pattern of left anterior hemiblock indicating disease of the anterior superior division of the left bundle. Each atrial depolarization, whether conducted or not, was followed by a His bundle potential. The most probable site of block in this patient was the distal segment of the His bundle. Another more unusual possibility would be simultaneous failure of conduction in the functioning right bundle and posterior inferior division of the left bundle. This concept is in agreement with the findings of Narula and coworkers (Narula and Samet, 1970; Narula *et al.*, 1971).

The His bundle electrograms in our second

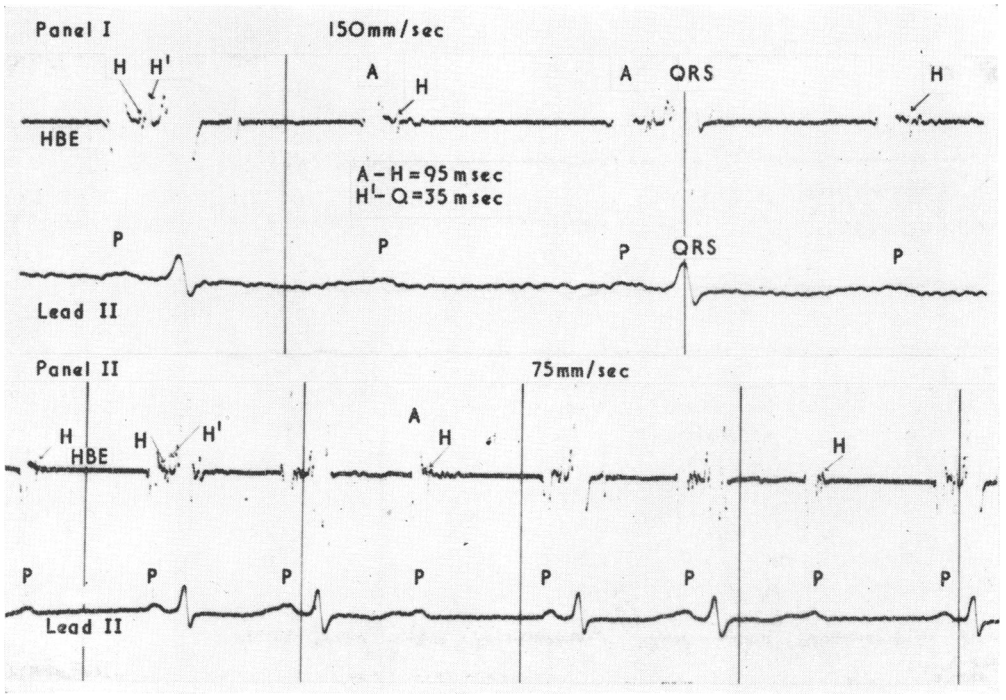


FIG. 4 *Case 2* (Panel I). *His bundle electrogram showing split His bundle with H and H' representing activity of proximal and distal His bundle segments, respectively. Block occurs following the H deflection.* (Panel II) *After intravenous atropine the AV conduction has improved from 2:1 to 3:2. The block again occurs distal to the H deflection.*

case clearly show block within the His bundle. Because of recent digitalis therapy and the presence of a normal QRS morphology, one is tempted to consider the AV node as the site of block. Intravenous atropine in this patient improved the AV conduction from 2:1 to 3:2. This is a rather unexpected finding in infranodal second-degree AV block. Haft, Weinstock, and DeGuia (1971) suggest that response to intravenous atropine may be of help in predicting the site of block from standard electrocardiograms. In the intranodal type of second-degree AV block, the conduction improves with atropine which is in contrast to block within the His bundle or bundle-branches where the dropped beats may increase. They have correlated this finding with the absence of autonomic control over the His bundle and bundle-branches.

Absence of response to atropine in our first case and a positive response in the second lead us to believe that the proximal His bundle may possibly be influenced by the autonomic nervous system. Since Narula and Samet (1970) did not report the action of atropine in their patients, we are unable to draw any definite conclusion from our limited experience.

Atrial pacing was performed in our second case when 1:1 AV conduction had returned. As expected, with increasing atrial rates the PI-H interval increased gradually (Damato *et al.*, 1969). However, Mobitz type II block within the His bundle (because HH' interval did not increase) was noted.

Electrophysiological findings in these two patients suggest that His bundle lesions may produce Mobitz type II AV block in the absence of bundle-branch block. Standard electrocardiograms are of no help in separating these patients from those with intranodal block. Atropine may improve conduction depending upon the site of delay within the His bundle. As pointed out by various investigators (Narula *et al.*, 1971; Rosen, Gunnar, and Rahimtoola, 1972), it is the site and not the pattern of block that should determine the prognosis and the need for pacemaker therapy. His bundle electrograms are necessary to localize the exact site of block, since predicting the site of block from the standard electrocardiogram may be misleading.

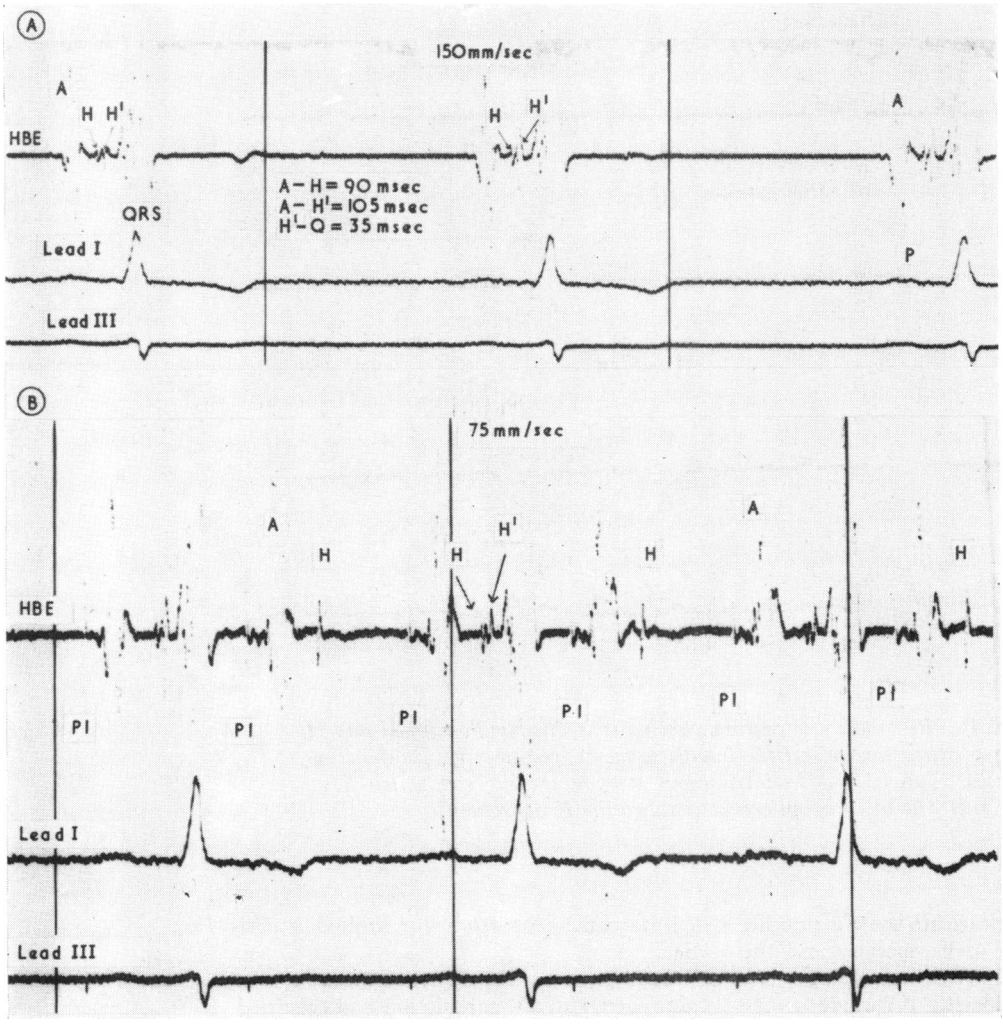


FIG. 5A *Case 2. His bundle electrogram recorded during 1:1 AV conduction. Split His activity is seen.*

FIG. 5B *2:1 AV conduction during atrial pacing at a rate of 150/minute. Block occurs distal to the H deflection.*

We are thankful to Dr. Jacob I. Haft for his helpful suggestions.

References

Damato, A. N., Lau, S. H., Helfant, R., Stein, E., Patton, R. D., Scherlag, B. J., and Berkowitz, W. D. (1969). A study of heart block in man using His bundle recordings. *Circulation*, **39**, 297.

Haft, J. I., Weinstock, M., and DeGuia, R. (1971). Electrophysiologic studies in Mobitz type II second degree heart block. *American Journal of Cardiology*, **27**, 682.

Langendorf, R., and Pick, A. (1968). Editorial. Atrioventricular block, type II (Mobitz) – Its nature and clinical significance. *Circulation*, **38**, 819.

Narula, O. S., and Samet, P. (1970). Wenckebach and Mobitz type II A-V block due to block within the His bundle and bundle branches. *Circulation*, **41**, 947.

Narula, O. S., Scherlag, B. J., Samet, P., and Javier, R. P. (1971). Atrioventricular block – localization and classification by His bundle recordings. *American Journal of Medicine*, **50**, 146.

Rosen, K. M., Gunnar, R. M., and Rahimtoola, S. H. (1972). Editorial. Site and type of second degree A-V block. *Chest*, **61**, 99.

Rosen, K. M., Loeb, H. S., Gunnar, R. M., and Rahimtoola, S. H. (1971). Mobitz type II block without bundle branch block. *Circulation*, **44**, 1111.

Rosen, K. M., Rahimtoola, S. H., and Gunnar, R. M. (1970). Pseudo A-V block secondary to premature

nonpropagated His bundle depolarizations: documentation by His bundle electrocardiography. *Circulation*, **42**, 367.

Scherlag, B. J., Lau, S. H., Helfant, R. H., Berkowitz, W. D., Stein, E., and Damato, A. N. (1969). Catheter technique for recording His bundle activity in man. *Circulation*, **39**, 13.

Spear, J. F., and Moore, E. N. (1971). Electrophysiologic studies on Mobitz type II second degree heart block. *Circulation*, **44**, 1087.

Requests for reprints to Dr. Prem K. Gupta, City Hospital Center at Elmhurst, 79–01 Broadway, Elmhurst, New York 11373, U.S.A.